

Amendments to the Claims

This listing of claims will replace all prior versions and listing of claims in the application:

CLAIMS

1 – 4. (Canceled)

5. (Currently amended) Animal model according to Claim ~~[[3]]~~ 16, characterized in that the mutation in the murine presenilin 1 gene comprising a M146L mutation-PS1 gene is replaced by or is in addition to a murine presenilin 1 gene mutation is selected from the group consisting of M146L, A246E, C410Y, H163R, L286V and L235P mutations, taken on their own or in combination.

6. (Canceled)

7. (Currently amended) Animal model according to Claim 16, characterized in that the mitochondrial dysfunction is an alteration, a modification, an overexpression or an inhibition of the expression of the mitochondrial proteins.

8. (Original) Animal model according to Claim 7, characterized in that the proteins are intramitochondrial proteins.

9. (Original) Model according to Claim 8, characterized in that the proteins are the Bax and/or cytochrome c proteins.

10. (Currently amended) A method for identifying compounds ~~which can be used for treating neurodegenerative diseases~~ that modulate the amyloid plaques, neuronal loss or mitochondrial dysfunction of the non-human transgenic animal model of claim 16 comprising exposing said compounds to the animal model ~~of any one of claims 1 to 9.~~

11 - 15. (Canceled)

16. (Currently amended) Non-human transgenic animal model that exhibits amyloid plaques, neuronal loss and mitochondrial dysfunction, said model comprising:

a nucleic acid sequence encoding a mutation in the murine presenilin 1 ~~protein~~ gene comprising a M146L mutation; and

a nucleic acid sequence encoding mutations in the human β -amyloid peptide protein precursor comprising the Swedish, Dutch and London mutations.